November 11, 1992

A NEW UNITED STATES CASE-CONTROL EPIDEMIOLOGIC STUDY OF ETS AND LUNG CANCER, THE LARGEST REPORTED TO DATE, REPORTS NO STATISTICALLY SIGNIFICANT ASSOCIATION

Ross Brownson, Michael ~~Alvanja~~ Alavanja, Edward Hock and Timothy Loy, Passive Smoking and Lung Cancer in Nonsmoking Women, American Journal of Public Health, 82, 11, 1525-1530, 1992

Overview

This is a large case control study of white non-smoking women from Missouri. It reports on 432 lifetime non-smoker cases, compared with 420 in the Fontham study, making the Brownson study the largest published case-control study ever conducted on this issue.

The study reports no statistically significant association between lung cancer and ETS exposure from a spouse, from all household members or ~~from~~ childhood ~~exposure~~. The relative risk reported for spousal smoke exposure and lung cancer is 1.0 with 95% confidence intervals of 0.8 to 1.2 -- that is, absolutely no increase in risk.

In spite of the data, the authors claim that the study indicates a small but consistent increased risk of lung cancer associated with ETS exposure and call for comprehensive actions to limit smoking in public places and worksites.

Claims made by the study

1. No increased risk of lung cancer was associated with childhood ETS exposure.

2. ~~Adulthood analyses showed~~ For adulthood exposure, the authors reported an increased lung cancer risk for lifetime nonsmokers with exposure of more than 40 pack-years from all household members (odds ratio 1.3, statistically not significant) or from spouses only (odds ratio 1.3, statistically not significant.)

3. This and other recent studies suggest a small but consistent increased risk of lung cancer from passive smoking.

4. Data on dietary intake of beta-carotene and fat was collected and was reported not to confound the data.

5. Comprehensive actions to limit smoking in public places and worksites are well advised.

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Talking points

1. This is the largest case-control study ever published. It reports no statistically significant association between lung cancer and ~~any surrogate for ETS exposure~~.

2. The relative risk for spousal smoke exposure and lung cancer is 1.0, indicating absolutely no increase in risk.

3. Although the authors fail to tabulate the data, they state that "[t]here was no elevated lung cancer risk associated with passive smoke exposure in the workplace."

4. The authors state that "[n]o increased risk of lung cancer was associated with childhood passive smoke exposure."

5. In fact, the odds ratios for childhood exposure are reported to decrease with increasing years of exposure.

6. The authors analyzed the data by lung cancer type. The odds ratio for spousal smoke exposure for adenocarcinoma was 1.0 (95% C.I. 0.8-1.3). In the highest exposure regime of over 40 cigarette pack years the odds ratio was 1.1 (95% C.I. 0.8-1.5). This data is entirely at odds with the claims made in the recently published Fontham study, where it was suggested that ETS exposure is specifically associated with adenocarcinoma.

7. Although the authors claim that the odds ratio of 1.3 reported for the highest exposure regime supports an association between ETS and lung cancer, there is absolutely no dose-response relationship in any of the data. For example, for spousal exposure and all lung cancer types the odds ratio for 0-15 cigarette pack years is 0.7, for 15-40 the odds ratio is 0.7, while only at >40 cigarette pack years does the odds ratio becomes 1.3.

8. The authors state "[w]hen the analyses were limited to direct interviews, no clear pattern of increase or decrease in risk estimates was apparent."

9. The authors examined the risks associated among women exposed in both childhood and adulthood, in childhood but not adulthood, and in adulthood but not childhood. They stated that, "[t]here was no evidence of interaction between exposure during the two periods." This again illustrates the lack of any dose-response relationship.

10. Brownson and co-workers collected information on age, active smoking (for ex-smokers), history of previous lung diseases, dietary beta-carotene and dietary fat. They stated that age, active smoking and previous lung disease all confounded the ETS data, and these factors were taken into

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account in the analyses. The dietary factors were, on initial analysis only, not reported to confound the data. The authors provided no information on what may be the most significant bias, misclassification.

11. The study reported results on both never smokers (432 cases) and ex-smokers (186). The ex-smokers were reported not to have smoked for at least 15 years prior to interview. The combined analysis of ex-smokers and never-smokers for spousal smoke exposure for more than 40 cigarette pack years and all lung cancers gave an odds ratio of 1.2 (95% C.I. 0.9-1.5), less than that when the analysis is restricted to never-smokers. This is not consistent with an effect from ETS exposure. claimed

12. The authors claim that the difference between their data and earlier studies (which reported associations between spousal smoke exposure and lung cancer) might be due to the decreasing prevalence of smoking among men. If this were true, then it would suggest that future studies will report no association between ETS exposure and lung cancer and that the older data ~~has~~ have little relevance to today's population.

13. Overall, the study ~~strongly supports~~ reports no significant lack of association between ETS exposure and lung cancer. The study is the largest of its type conducted to date, and adds considerable weight-of-evidence ~~to~~ for a conclusion that ETS exposure does not increase the risk of lung cancer.

(quantitative standards)

Covington and Burling

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